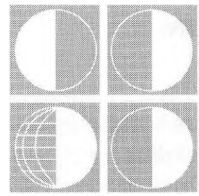
Fertility and Post-Reproductive Longevity



Ken R. Smith[a,d], Geraldine P. Mineau[b,d], and Lee L. Bean[c]

[a]Department of Family and Consumer Studies, 225 South 1400 East, Rm 228, University of Utah, Salt Lake City, Utah 84112, ken.smith@fcs.utah.edu; [b]Department of Oncological Sciences, 2000 Circle of Hope, University of Utah, Salt Lake City, Utah 84112-0900, geri.mineau@hci.utah.edu; [c]Department of Sociology, 301 Social Behavioral Science Building, University of Utah, Salt Lake City, Utah 84112, bean@freud.sbs.utah.edu; [d]Population Sciences, Huntsman Cancer Institute, 2000 Circle of Hope, University of Utah, Salt Lake City, Utah 84112-0900

ABSTRACT: **We examine the effects of reproduction on longevity among mothers and fathers after age 60. This study is motivated by evolutionary theories of aging and theories predicting social benefits and costs of children to older parents. We use the Utah Population Database, that includes a large genealogical database from the Utah Family History Library. Cox proportional hazard models based on 13,987 couples married between 1860–1899 indicate that women with fewer children as well as those bearing children late in life live longer post-reproductive lives. As the burdens of motherhood increase, the relative gains in longevity of late fertile women increase compared to their non-late fertile counterparts. Husbands' longevity is less sensitive to reproductive history, although husbands have effects that are similar to those of their wives during the latter marriage cohort. We find some support for predictions based on evolutionary principles, but we also find evidence that implicates a role for shared marital environments.**

INTRODUCTION

Childbearing and child rearing affect the lives of parents. It is well known that childbirth has significant health effects on mothers during their childbearing years (National Research Council, 1989). While numerous studies demonstrate the effects of female reproductive history on cancer and heart disease mortality, far less is known about the influences of fertility patterns on the longevity of mothers and fathers who survive their reproductive years. Interest in this question has increased recently as a result of work by evolutionary biologists and biodemographers (Westendorf and Kirkwood, 1998; Rose, 1997; Vaupel et al., 1998). In describing the field of biodemography and its links to evolutionary concepts, Vaupel et al., (1998) recently noted that "It is reproductive success that is optimized [through natural selection], not longevity. Deeper understanding of survival at older ages thus hinges on intensified research into the interactions between fertility and longevity." We suggest, therefore, that it is important for demographers to examine the association between fertility history and post-reproductive aging, particularly from the perspective of both evolutionary and social theories (Wachter and Finch, 1997).

We revisit the issue of fertility and post-reproductive longevity in light of two recent developments. First, evolutionary biologists and biodemographers have recently argued that female fertility patterns in various species, including humans, may be useful markers for rates of biological aging (Kirkwood and Rose, 1991; Vaupel et al., 1998). A reexamination of the association between human female fertility and longevity is therefore warranted based on evolutionary theories of aging. Second, family demographers

and sociologists have long argued that children may serve as assets (especially in agricultural and pre-industrial settings) when children are young and, as adults, these children may also provide social and economic support to their middle-aged and elderly parents (Wolf, 1994). The presence of children may therefore play an important role in providing health-enhancing social support to their older parents. In an effort to evaluate the contributions stemming from both theoretical orientations (which yield similar predictions in some cases while differing in others), we examine the effects of childbearing and child survival on the longevity of mothers and fathers.

There are few sources of information that provide extensive reproductive as well as survival information across the entire life course of mothers and fathers for a well-defined population. A promising approach for the study of fertility and parental longevity has been to use genealogies. Many of the earliest fertility-longevity studies using such data relied on small samples or selected family lineages. We employ the Utah Population Database (UPDB), a large, heterogeneous genealogical database. The UPDB is derived from records maintained by the Utah Family History Library in the Church of Jesus Christ of Latter-day Saints (LDS or Mormon). These records have been enhanced through links to vital records and for many years span a period of history where natural fertility conditions prevail.

BACKGROUND

Early Studies of Fertility and Post-Reproductive Longevity

The first scientific investigations of female fertility and post-reproductive longevity occurred at the turn of the century (Beeton et al., 1900; Powys, 1905; Bell, 1918). Freeman (1935) and Dorn and McDowell (1939) later considered the positive association between female fertility and longevity and confirmed these earlier findings that the average number of children born was positively associated with age at death among women. Neither report offered *a priori* predictions about the positive fertility-longevity association except to suggest a form of positive selection for women: initially robust women have more children and robust women live longer. They also acknowledged, however, that increasing fertility may place a "tax on the vitality" of women who bear many children which might lead to a negative association between fertility and longevity. These earlier studies examined the association between completed fertility and longevity but did not consider other aspects of fertility such as age at first and last birth, nor did they consider how reproduction may affect male longevity.

Fertility, Longevity, and Evolutionary Biology

Demographers have only recently introduced perspectives from evolutionary biology in their analyses of aging and mortality (Carnes and Olshansky, 1993; Wachter and Finch, 1997). A number of interesting research issues have been stimulated by such work. Wachter (1997), for example, argued that demographers may be motivated to use evolutionary concepts to enhance their understanding of the biological limits of longevity as developed societies appear to be approaching human life-span maximums.

Evolutionary biology provides a potentially useful addition to demographic analyses of the inter-relationships between

fertility and longevity. We introduce a brief set of fundamental ideas within evolutionary biology. Evolutionary theory argues that there are trade-offs that each organism makes between investing resources into somatic or physical growth and investing in reproduction (Kirkwood, 1977; Lycett et al., 2000). The general problem is framed as an optimization issue where each species is trying to maximize their reproductive success within the species' prevailing mortality constraints. The idea of a trade-off between reproduction and longevity is called the disposable soma theory (Kirkwood, 1977; Kirkwood and Rose, 1991). This theory predicts that, for females, young age at first birth and high parity will be associated with a shorter post-reproductive life span because early or high levels of fertility exact high physical (somatic) costs to such mothers which, in turn, shorten their lives.

Why might such trade-offs exist? Williams' classic work (1957) advanced an antagonistic pleiotropy theory of senescence to address this question. Pleiotropy refers to genes that have more than one function such as having one function at one age and another at a later age. In particular, genes that enhance reproductive capacity will be preserved through natural selection but these same genes may accumulate at later ages, perhaps proving to be deleterious to post-reproductive survival. If deleterious effects of such genes occur late in life (i.e., post-menopausal) and the beneficial effects occurring early in life are favored by natural selection, then survival may be determined by selection for fertility-related traits. This theory also predicts that early and higher levels of fertility should be associated with shorter life spans of mothers, a prediction consistent with disposable soma theory.

As these evolutionary concepts suggest, genes deleterious to reproduction will be eliminated through natural selection and, given the inability of natural selection to eliminate late-acting (post-menopausal) deleterious genes, we should see an increase in mortality rates following the cessation of reproduction as measured by age at natural menopause or age at last birth. Several evolutionary scientists (Hamilton, 1966; Charlesworth, 1980; Kirkwood and Rose, 1991) have therefore theorized that forces that prolong the period during which female reproduction occurs will postpone aging and increase female longevity. This line of reasoning suggests that increasing ages at last birth among females should be associated with greater post-reproductive female longevity.

This body of work leads to three specific hypotheses:

H1: As age at first birth increases, post-reproductive parental longevity increases.

H2: As the number of children decreases, post-reproductive parental longevity increases.

H3: As the age at last birth increases, post-reproductive parental longevity increases.

The empirical evidence on humans is mixed for these predictions. Westendorf and Kirkwood (1998) reported that women listed in genealogies of the British aristocracy who survived to age 60 died earlier if they had higher parity and a younger age at first birth compared to women with fewer children and later ages at first birth. With contemporary data from England and Wales (1971–96) and Austria (1981–1982), Doblhammer (2000) reported that women lived somewhat longer if they had few children and

if their age at first birth occurred after age 20. Friedlander (1996) found that parous women had lower survivorship than nulliparous women, and among parous women, those with higher parity lived shorter lives than those with lower parity. Beral (1985) reported that parous women had lower overall survival than nulliparous women, primarily because of an elevated risk of circulatory diseases, findings consistent with those of Kvale et al., (1994). Conversely, some investigators (Lund et al., 1990; Green et al., 1988) reported that nulliparous women have lower survival than their parous counterparts. Bideau (1986) concluded that women with extreme fertility (more than twelve children) enjoyed the best survival. Using data from the population register of Canadians in the seventeenth and eighteenth centuries, Le Bourg et al. (1993) could find no support for the predicted trade-offs between early "fecundity" (i.e., fertility) and later survival. In general, investigations on the trade-offs between parity and post-reproductive longevity have not simultaneously considered the effects of late fertility.

A positive association has been reported between late female fertility (Perls et al., 1997; Doblhammer, 2000) or late menopause (Snowdon et al., 1989; Cooper and Sandler, 1998) and longevity. Another analysis found no such association after controlling for the effects of smoking history and socioeconomic status (Egan et al., 1997). Isolating the hypothesized longevity benefits attributable to late female fertility in humans is complicated by the fact that women bearing children at very late ages usually have higher parity when natural fertility patterns prevail. The few studies that have considered the longevity benefits of late female fertility have not systematically analyzed the competing influences of high parity (see Snowdon et al., 1989, for an exception).

Effects on Parents of Social Support and the Cost of Children

Studies of fertility and post-reproductive longevity have often examined the role that childbearing plays in hormonal or evolutionary terms. Less attention has been given to the possibility that social relationships between adult children and parents may influence parental post-reproductive longevity. It is well established that individuals with greater access to social support have better health and lower levels of mortality (House, Landis, and Umberson, 1988; Ross, Mirowsky, and Goldsteen, 1990). After marriage partners, children are generally regarded as the most important component of an adult's social and family network (Lye, 1996; Logan and Spitze, 1996; Wolf, 1994), although it is unclear whether adult children affect the longevity of their middle-aged and elderly parents. Adult children may affect the longevity of their parents but primarily when parents are seriously ill or elderly (Mancini and Blieszner, 1989; Silverstein and Bengston, 1991), particularly when adult daughters are present (Pearlin et al., 1995).

Assistance and resource exchange between adult children and their parents flow in both directions but largely migrate from parents to children. When the flow of resources (social support and income) moves from children to parents, it is small for contemporary U.S. families (Hogan et al., 1993) as well as for preindustrial societies (Lee, 1997; Kaplan, 1994). If children receive more resources than they provide to their parents, then

the "cost" of children to parents may serve to reduce parental longevity.

Are adult children constrained in their capacity to provide support for their aging parents? These adult children are themselves rearing offspring of their own. In this three-generational scenario, grandchildren may serve to reduce the ability of children to provide support to grandparents. Given that fertility patterns are "transmitted" across generations (Anderton et al., 1987), higher-parity parents will be more apt to have children who themselves will have higher parity, thereby limiting the children's capacity to provide assistance to their parents. This argument suggests that during periods of natural fertility, parents with high parity will be adversely rather than beneficially affected since their own high-parity children will be devoting resources to their own childrearing.

Parents bearing their first children at younger ages are more likely to invest their limited economic resources to child rearing rather than to their own personal health, employment, or savings (Waldron et al., 1998; Hofferth, 1984). This logic suggests that individuals bearing children later in life, with other things being equal, would also experience adverse health consequences given the extended period of time over which the demands of child rearing would accumulate.

These social mechanisms suggest the following hypotheses:

H4: As age at first birth increases, post-reproductive parental longevity increases.

H5A: As the number of children increases, post-reproductive longevity *increases* (due to access to greater informal social support).

H5B: As the number of children increases, post-reproductive longevity *decreases* (due to wealth flows largely moving from parent to child; children are also high-parity themselves, thereby reducing their supportive capabilities).

H6: As the age at last birth increases, post-reproductive parental longevity decreases.

Hypotheses H1 and H4 (age at first birth) as well as H2 and H5B (parity) make the same predictions although they invoke different mechanisms. Hypotheses H2 and H5A (parity) conflict as do hypotheses H3 and H6 (late age at last birth). Accordingly, we are better able to assess the underlying forces that link longevity with either parity or late age at last birth than we are with respect to age at first birth.

Possible Confounding and Selection Mechanisms

Alternative processes may give rise to an association between fertility and post-reproductive longevity. We consider the role of initial health status, child mortality, religion, socioeconomic status (SES), and secular trends in fertility and mortality.

Some early investigators (Prows, 1905; Dorn and McDowell, 1939) suggested that individuals have health characteristics that increase their chances of marriage, coital frequency, and bearing children, as well as reducing their risks of mortality. This position suggests that some women are more robust, leading to greater fecundity and longevity (see Samuelsson and Dehlin, 1993). We do not possess initial health status measures in our data to directly address this issue. This argument will be persuasive only if

all of our fertility measures presumed to be associated with greater vitality and health (high parity, early age at first birth, late age at last birth) lead to an extended life span.

The role of fertility on post-reproductive longevity may be confounded when childhood mortality rates are high. Two couples with identical parity may be very different with respect to the number of surviving children and hence the effects of childrearing on subsequent parental mortality could also be very different. In addition, child deaths may be a marker for adverse environments experienced by mothers and hence can be viewed as a measure of early adult exposures that affect both parental mortality and fertility. We therefore include as a covariate the number of children who died before age 18 for a given couple.

Religious affiliation may also promote an association between fertility and longevity. Couples committed to the LDS church are more likely to live longer (Enstrom, 1978, 1989) and to have more children (Bean, Mineau, and Anderton, 1990) than other couples. While the population represented in the UPDB are genetically representative of western European populations, active Mormons generally live longer because church-related activities and doctrine enhance social cohesion and serve to reduce alcohol and tobacco consumption (Bush, 1993). For these reasons, we include a measure of the strength of LDS commitment in our analysis.

A family's SES may also encourage an association between fertility and longevity. High-status marriages are more likely to experience lower levels of fertility and mortality. The role of social status makes sense only if fertility was somehow controllable during the late nineteenth and early twentieth centuries. Morgan (1991) found that methods of fertility limitation were in use during this time, although his analysis did not specifically consider Mormon fertility. Nonetheless, after controlling for marital duration and age at marriage, Morgan (1991:795–97) showed that women with professional spouses had lower fertility than farming wives based on data from the 1910 U.S. Census. At the same time, we know that high SES individuals live longer than their lower-status counterparts (Antonovsky, 1967; Kitagawa and Hauser, 1973; Townsend et al., 1988; Smith and Waitzman, 1994; McIntyre, 1997). For women married in the latter half of the 1800s, their status is best represented by their husbands' SES; accordingly, we rely on husbands' SES in this analysis.

Most families in Utah were engaged in agriculture during the last half of the nineteenth century. This is relevant given that farm couples had a preference for large families, particularly given the high rates of infant mortality at that time. During the latter portion of this period, an urban core was developing in what is now the Salt Lake City–Ogden–Provo MSA, leading to declines in the proportion of farm families. This trend offers an opportunity to assess whether female reproductive patterns have varying effects on longevity as the value of large families changed over time.

Finally, secular trends in fertility and mortality are considered. Our investigation spans an historical period that includes patterns of natural fertility for the majority in the Utah population during the late nineteenth and early twentieth century (Bean et al., 1990). Couples marrying from 1860–74 make up the earliest period in this study and reflect a period of natural fertility on a frontier, while the

marriage cohort of 1875–99 represents the beginning of the fertility transition. In general, individuals married between 1860 and 1874 would be exposed to more hardships associated with migration to the West, uncertain food supplies, limited medical care, and physical hazards. Those married later in the nineteenth century (1875–99) were more likely to have been born in Utah and have experienced fewer hardships as transportation and a developing infrastructure in the West enhanced the quality of life and reduced the risks of mortality.

DATA

We examine our hypotheses using the genealogical data within the UPDB. The UPDB was developed for medical research, and since 1974 numerous enhancements have been made, including vital status information from Utah Death Certificates and the Health Care Financing Administration (HCFA). The UPDB is based on information from over one million individuals representing about 185,000 "Family Group Sheets" abstracted from the Utah Family History Library. Because these records include basic demographic and religious data on parents and their children, we can measure for each person their fertility history, age at death (or age last known alive), and religious affiliation with the LDS church.

From the UPDB, 13,897 couples have met the following selection criteria and comprise our analysis sample. Monogamous couples were selected who married between 1860 and 1899. This time period was used to identify couples who were most likely to have married in Utah and who would likely have observed death dates. This time interval generally excludes childbearing during the well-known fertility decline of the Great Depression. Overall, the UPDB data on marriages established during the latter half of the nineteenth century are particularly advantageous for testing both social and evolutionary theories, given that fertility control was limited and mortality risks later in life were relatively high.

Only once-married couples were included to limit complications related to fertility spanning more than one marriage partner. Husbands could not be more than ten years younger nor fifteen years older than their wives to reduce large differences in age and cohort experiences.

Wives were required to have married no later than their thirty-fifth birthday to ensure that they had a clear opportunity to bear children. All wives selected for the analysis lived to age 60 to assure that all women would have completed their childbearing. Couples with husbands fathering children past age 60 were also excluded. Some wives who lived to age 60 were already widowed. We selected those couples whose husbands were alive when their wives reached 60 years of age. Husbands who died before their wives' sixtieth birthday would have left wives without a crucial source of social support and, for younger husband deaths, an inability to bear children (if no remarriage occured). Early or midlife widowhood could, therefore, lead to a reduction in both fertility and female life span.

Divorced couples are omitted given that divorce was exceptionally rare during the period covered by this analysis. Polygynous marriages were also excluded from consideration because they introduce an added level of complexity that applies only to a small percentage of privileged men (under 10 percent) concentrated during the earliest marriage cohort (Arrington,

1959; Bean et al., 1987). Consanguineous marriages, though rare, are also excluded in order to remove the remote possibility that the children of such unions would be at an increased risk of death (Bittles et al., 1991). Couples whose first child was born out of wedlock were excluded although couples whose first child was born six months or later after the marriage were included to allow for premature births. Nulliparous marriages are not used in the analyses as they comprise less than one percent of all marriages between 1860 and 1899. The analysis includes the effects of age at first birth on the parents' mortality risk, making it necessary to restrict the sample to parous couples. Nulliparous marriages may be slightly underreported in the UPDB, as they are in most genealogies, since no descendants exist to provide family data. On the other hand, men and women in nulliparous marriages are typically represented because they themselves are part of sibships that are reported quite well in the UPDB.

Death dates are available from the genealogy, Utah death certificates (1934–1992) linked to the UPDB, and for decedents identified through files maintained by Health Care Financing Administration. No men and only 14 women were known to be alive in 1997 among persons married between 1860–99.

SURVIVAL ANALYSIS METHODS

Cox proportional hazard rate models are used to estimate the survival models. The parametric distribution of the baseline hazard $h_{0i}(t)$ is left unspecified although different for husbands and wives:

$$(1) \qquad h_{ij}(t) = h_{0i}(t)\exp(b\, z_{ij}),$$
$$i = 1 \text{ for wives, } i = 2 \text{ for husbands,}$$

where $h_{0i}(t)$ is a sex-specific baseline hazard, z_{ij} are *observable* covariates for the ith spouse in the jth couple, and b is a vector of regression parameters. The hazard rate is assessed based on mortality after age 60.

This specification assumes that there are no significant unobservable couple-specific effects after controlling for observable independent variables. Previous analyses of these data (Smith, Mineau, Guo, and Huth, 1995) found small and insignificant unobservable couple-specific effects when such (random) effects followed a gamma distribution (Guo, 1993; Guo and Grummer-Strawn, 1993).

We also consider how observable indicators of fertility affect mortality risks differently for a mother and a father in the same marriage. We use pair-rank models to test for such differences (Holt and Prentice, 1974; Smith and Zick, 1994; Smith and McClean, 1998) in the presence or absence of significant unobserved heterogeneity. For the pair-rank model, we modify equation 1 as follows:

$$(2) \qquad h_{ij}(t) = h_0(t)\exp(b\, z_{ij})w_j$$
$$= h_{0j}(t)\exp(b\, z_{ij})$$

where $h_{0j}(t) = h_0(t)w_j$ and w_j is a couple-specific indicator for unobserved heterogeneity. The baseline hazard function $h_{0j}(t)$ is the hazard rate that is shared by both spouses of the jth couple and its distribution need not be specified (Huster et al., 1989; Kalbfleisch and Prentice, 1980; Oakes, 1986; Wild, 1983).

One can obtain estimates of b from equation 2 by using logistic regressions for the within-pair rank of survival time that removes the effects of shared unobserved heterogeneity (Kalbfleisch and Prentice, 1980; Smith and McClean,

1998). This is done by taking the ratio of the jth wife's and husband's hazard rate equations. Consider, for example, whether the effect of parity, $Z_{parity,j}$, is different for wives and husbands from couple j:

$$(3)\ h_{1j}(t)/h_{2j}(t) = \{h_{0j}(t)\exp(b_W * z_{parity,j})\} \div \{h_{0j}(t)\exp(b_H * z_{parity,j})\} = \exp(b_W * z_{parity,j})/\exp(b_H * z_{parity,j}) = \exp[(b_W - b_H) * z_{parity,j}]$$

A logistic regression can be used to estimate $(b_W - b_H)$:

$$(4)\ \text{logit}\,(r_j) = b_0 + ((b_W - b_H) * z_{parity,j})$$

where the dependent variable, r_j, equals 1 if the wife's age at death (minus one) from couple j is lower than her husband's and 0 otherwise (Kalbfleisch and Prentice, 1980). In these data, women outlive husbands by one year given survival to age 60. To take this gender difference in survival, we make this one-year adjustment in the construction of r_j. In a pair-rank model, the null hypothesis states that the effect of parity is the same for a wife and husband (H_0 is $(b_W - b_H) = 0$). A single regression coefficient $\Delta b = b_W - b_H$ is estimated for $z_{parity,j}$ that quantifies the difference in the effect that parity has on the wife's and husband's mortality risk.

MEASURES

The covariates included in each Cox and pair-rank regression include dummy variables for year of marriage, wife's age at first birth, total number of children, wife's age at last birth, and total number of children that died before they reached age 18, and a dummy variable indicating whether the wife is LDS or not. Wife's age at first birth, age at last birth, and religious status were strongly correlated with the husband's values of these measures. We have elected to use the wife's values in both husband and wife equations. This approach also has the advantage of making hypothesis testing for differences in effects between spouses straightforward in pair-rank regressions. Wife's age at marriage is not used as it is strongly correlated with wife's age at first birth (zero-order Pearson correlation is 0.96). Number of children, year of marriage, age difference between spouses, and number of deceased minor children are couple-level variables by definition.

Religious commitment is based on a classification scheme developed by Mineau (1980) and Bean et al. (1983, 1990) using the timing of an important religious rite, endowment, in the LDS church. Endowment, a pledge to the LDS church and its doctrine, usually takes place early in adulthood, before an individual goes on an LDS mission, at the time of a "temple" marriage, or later for converts and reactivated members. Individuals who have records containing endowment dates before age forty are treated as religiously committed to the LDS church. Others are less committed or non-LDS members.

For fertility variables, both continuous and categorical specifications were implemented. For the categorical versions, the middle 50 percent of the distribution was used as the excluded category and four dummy variables were constructed that corresponded to the following percentiles: below the 10th, between the 10th and 25th, between the 75th and 90th, and above the 90th. For wife's age at last birth, we further divided the highest percentile category into two categories:

between 90th and 95th and above the 95th percentiles.

For a portion of the sample, occupational data are available. Analyses incorporating occupational information are limited to marriages from the 1875–99 marriage cohort, where the linkage rate between UPDB records and death certificates was 67 percent. The linkage rate was lower (45 percent) for the earlier marriage cohort. Occupations were coded to the 1980 U.S. Census categories and assigned a socioeconomic status score based on Nam and Powers (1983). Higher scores represent occupations with greater socioeconomic status. If a husband had no Utah death certificate or one that did not link to the UPDB, he was included in the sample and was assigned the mean Nam-Power SES score. An additional variable was then added to the model that indicated whether or not the husband had missing SES information to adjust for the effects of this mean imputation. Over 50 percent of husbands with known occupations were employed in farming. Accordingly, a farming dummy variable was introduced into the model in order to distinguish the effects of farming from all other occupations encompassed by the Nam-Power status score.

RESULTS

The findings reported below are based on multivariate models that include measures of age at first and last birth, parity, year of marriage, age difference between spouses, mortality of offspring, and commitment to the LDS church (Table 1). Mean age at first birth was 21.73 for wives and 25.58 for husbands. Wives gave birth to their last child at a mean age of 40. Families averaged 8.24 children with 1.3 dying before the children reached age 18. Nearly 70 percent of couples were committed to the LDS church.

Table 2 lists results for several Cox proportional hazards models for female mortality. All covariates are measured as continuous variables. When each of the three reproductive history variables are included separately (Models 1–3), only parity affects female survival ($p < 0.10$). However, the simultaneous inclusion of all three fertility measures reveals strong influences of parity and wife's age at last birth (Model 4). Decreasing parity and increasing age at last birth are associated with a decreasing risk of female mortality past the age of 60. Age at first birth is not significantly associated with the mortality hazard rate. These three fertility measures are correlated with one another (e.g., simple Pearson correlations are: $R_{(\text{age at first birth, age at last birth})} = +0.07$, $R_{(\text{age at first birth, parity})} = -(0.42)$, $R_{(\text{age at last birth, parity})} = +0.64$), but do not represent any serious collinearity problems when introduced simultaneously into a model.

We next consider the additional effects of interactions between each of the three fertility measures (Models 5–8). All covariates involved in interactions have been centered to reduce collinearity problems (e.g., inflated standard errors of the regression coefficients) induced by the inclusion of multiplicative interaction terms. Model 7 includes the only statistically significant interaction, that involving age at last birth and parity. This interaction is negative, indicating that the reduction in mortality risk associated with late fertility is greatest at higher levels of parity. These results suggest that late fertile women are most clearly differentiated from other women when the physiological and social demands of reproduction and child rearing are greatest: when parity is high. It is possible that the interaction between par-

TABLE 1

DESCRIPTIVE STATISTICS (N = 13,897 COUPLES)

	MEAN	SD	MINIMUM	MAXIMUM
Wife's years lived since age 60	23.30	9.31	0.01	50
Husband's years lived since age 60	22.14	8.62	0.01	45
Marriage date	1884.62	10.39	1860.00	1899.99
Wife is LDS (= 1)	0.69	0.46	0	1
Husband is LDS (= 1)	0.68	0.47	0	1
Wife's age at first birth	21.73	3.29	15.66	38.30
Husband's age at first birth	25.58	3.80	16.83	49.35
Wife's age at last birth	39.99	4.64	18.65	54.94
Husband's age at last birth	43.83	5.66	20.24	59.98
Parity (all couples are parous)	8.24	2.85	1	19
Husband-wife age difference	3.84	3.65	−9.87	14.95
Number of children who died before age 18	1.33	1.41	0	11
Nam-power occupational ranking based on husband's death certificate information (for marriages 1875–1899)	44.78	15.87	3	99
Farmer (= 1) based on husband's death certificate information (for marriages 1875–1899)	0.59	0.49	0	1
Whether married 1860–1874 (= 1)	0.20	0.40	0	1

ity and age at last birth may simply represent the effects of birth intervals. High-parity women with a young age at last birth will have shorter birth intervals than high-parity women with an older age at last birth. Including birth spacing as a main effect resulted in no significant effects on the mortality risk of mothers (results not shown).

Cox regression models were re-estimated with the three fertility indicators included as categorical variables (Table 3). Model 4 includes all three sets of fertility variables simultaneously in a Cox regression, while Model 5 is a re-estimation of Model 4 except that we use a logistic regression where the outcome is whether or not a woman reaches age 95. In Model 4, we find that women with few children (1 to 3) have the lowest mortality risk relative to modal women with 7 to 11 children. These same low-parity women have a 63 percent greater chance of reaching age 95 than women from the middle 50 percent of the parity distribution (Model 5). Mothers whose last birth occurred at age 46.5 or later (i.e., beyond the 95th percentile for the age-at-last-birth distribution) have a mortality hazard rate that is 15 percent lower than that of women whose last child was born between ages 37.7 and 43.1 (i.e., the middle 50 percent of the age-at-last birth distribution). These late fertile mothers have a 44 percent greater chance of reaching age 95 than comparison mothers (Model 5).

We next divided the sample into two broad marriage cohorts and introduced analyses of husbands' survival. These two modifications were used to assess the effects of reproductive history on male and female longevity and how this association

TABLE 2

WIVES' HAZARD RATE MODELS FOR SURVIVAL PAST AGE 60. ENTRIES ARE COX PROPORTIONAL HAZARD REGRESSION COEFFICIENTS MULTIPLIED BY 10^3. ALL COVARIATES ARE CONTINUOUS VARIABLES.

VARIABLE	MODEL 1	MODEL 2	MODEL 3	MODEL 4	MODEL 5	MODEL 6	MODEL 7	MODEL 8
MAIN EFFECTS								
Age at first birth	−2.9	—	—	4.17	4.53	4.22	4.35	4.68
Parity	—	5.7*	—	18.2***	18.4***	18.3***	19.4***	19.5***
Age at last birth	—	—	−2.5	−9.05***	−9.06***	−8.96***	−12.6***	−12.6***
2-WAY INTERACTIONS								
Age at last birth * parity	—	—	—	—	0.27	—	—	0.26
Age at first birth * age at last birth	—	—	—	—	—	0.24	—	−0.074
Age at last birth * parity	—	—	—	—	—	—	−1.56***	−1.57***
Δ -2LL (Model-2LL vs. Null-2LL)	86.71	88.41	87.46	99.26	99.35	99.42	105.85	105.92
Δ df (Model df vs. Null df)	7	7	7	9	10	10	10	12

Adjusts for marriage year, LDS commitment, age difference between spouses, and number of children who died before age 18.
*$p < 0.10$, **$p < 0.05$, ***$p < 0.01$

TABLE 3

WIVES' HAZARD RATE MODELS FOR SURVIVAL PAST AGE 60. ENTRIES ARE RATE RATIOS BASED ON COX PROPORTIONAL HAZARD REGRESSION COEFFICIENTS FOR MODELS 1–4. MODEL 5 IS BASED ON A LOGISTIC REGRESSION PREDICTING THE ODDS OF LIVING TO AGE 95. ALL FERTILITY COVARIATES ARE CATEGORICAL.

VARIABLE		MODEL 1	MODEL 2	MODEL 3	MODEL 4	MODEL 5
AGE AT FIRST BIRTH						
Percentile (Age ranges)						
< 10th	(< 18.12)	1.00	—	—	0.98	1.01
10th–24th	(18.12–19.38)	1.03	—	—	1.01	1.02
25th–74th	(19.39–23.39)	1.00	—	—	1.00	1.00
75th–89th	(23.40–26.07)	0.98	—	—	0.99	1.02
> = 90th	(> = 26.08)	1.01	—	—	1.05	0.86
PARITY						
Percentile (Number of children)						
< 10th	(1 to 3)	—	0.93*	—	0.86***	1.63***
10th–24th	(4 to 6)	—	0.99	—	0.95**	1.18**
25th–74th	(7 to 11)	—	1.00	—	1.00	1.00
75th–89th	(12 to 14)	—	1.03	—	1.05*	0.96
> = 90th	(≥ 15)	—	1.08	—	1.13	0.92
AGE AT LAST BIRTH						
Percentile (Age ranges)						
< 10th	(< 33.3)	—	—	1.02	1.10**	0.76**
10th–24th	(33.3–37.6)	—	—	1.02	1.05*	0.92
25th–74th	(37.7–43.0)	—	—	1.00	1.00	1.00
75th–89th	(43.1–44.8)	—	—	1.02	1.01	0.99
90th–94th	(44.9–46.4)	—	—	0.96	0.93*	1.07
> = 95th	(> = 46.5)	—	—	0.87***	0.85***	1.44**
Δ -2LL		88.05	90.15	98.48	114.62	74.76
Δ df		10	10	10	19	19

*$p < 0.10$, **$p < 0.05$, ***$p < 0.01$

Adjusts for marriage year, LDS commitment, age difference between spouses, and number of children who died before age 18.

changed with time. Models 1 and 3 in Table 4 list the effects of reproductive history on survival for wives and husbands, respectively, for the 1860–74 marriage cohort. In general, women who had few children as well as those who were fertile late had lower rates of mortality, a pattern that is not observed among husbands.

When wives from the 1860–74 cohort are matched to their husbands and a pair-rank regression is estimated (Model 5), we find that the longevity gains associated with late female fertility are somewhat greater for wives than for their husbands (although not significant, {p = 0.20}, suggesting that the negative [protective] coefficient for wives is somewhat larger than it is for husbands). Similarly, the adverse effects of high parity are larger for wives than they are for husbands ($p < 0.05$).

We observe a different pattern of survival for couples from the 1875–1899 marriage cohort (Models 2 and 4). The effects of parity, late age at last birth, and their interaction persist among women, but we now observe a significant protective effect of late fertility among husbands. While the mortality risks of wives continue to be affected by their fertility

TABLE 4

WIVES' AND HUSBANDS' HAZARD RATE MODELS FOR SURVIVAL PAST AGE 60, BY TWO MARRIAGE COHORTS. ENTRIES FOR MODELS 1–4 ARE COX PROPORTIONAL HAZARD REGRESSION. ENTRIES FOR MODELS 5 AND 6 ARE LOGISTIC REGRESSION COEFFICIENTS BASED ON THE PAIR-RANK MODEL. ALL COEFFICIENTS ARE MULTIPLIED BY 10^3.

	WIVES		HUSBANDS		WIFE–HUSBAND COMPARISONS (PAIR-RANK MODELS)	
VARIABLE	Model 1 Married 1860 74	Model 2 Married 1875 99	Model 3 Married 1860–74	Model 4 Married 1875–99	Model 5 Married 1860–74	Model 6 Married 1875 99
Age at first birth	10.9	2.7	−21.1***	−7.75*	10.3	11.8
Parity	33.1***	16.2***	−15.9	1.39	53.7**	10.2
Age at last birth	−19.7***	−10.9***	−5.35	−9.77**	−17.0$^{\&}$	−0.74
2-WAY INTERACTION						
Age at last birth * parity	−2.1$^{\&}$	−1.4**	−0.17	−0.85	−3.55$^{\&}$	−1.14
N	2757	11126	2757	11126	2757	11126
Δ -2LL	12.13	58.80	27.39	120.47	17.68	61.31
Δ df	8	9	8	9	8	9

$^{\&}p < 0.20$, $^{*}p < 0.10$, $^{**}p < 0.05$, $^{***}p < 0.01$

Adjusts for marriage year, LDS commitment, age difference between spouses, and number of children who died before age 18.

Regression coefficients (b) are based on spouse-specific Cox proportional hazard rate models. Pair-rank tests are estimated with binary logistic regression and estimate the differences in effects between spouses (wife–husband) in the same marriage controlling for shared unobserved heterogeneity.

history, their effect sizes are attenuated relative to the previous cohort. To the extent that couples were beginning to limit their family size during this period relative to the preceding fifteen years, it may be that some women who could have had children at older ages or could have had an additional child did not do so due to greater use of available methods of fertility control. If this was the case, then some fecund women were categorized as having lower parity or having a lower age at last birth, both of which would reduce the estimated survival effects of parity and late fertility. At the same time, the effect of age at last birth on husband's mortality in this latter cohort increased. One possible explanation for this is that previously high rates of non-aging-related male mortality past age 60 were declining, thereby allowing the slow aging effects suggested by male late fertility to be better observed. Based on pair-rank methods for this later marriage cohort, a couple's history of childbearing yields similar longevity patterns for wives and husbands.

We find no evidence that age at first birth affects female mortality, but it does affect male mortality. Men with wives whose first child is born at later ages experience greater survival relative to other men, a result observed for both marriage cohorts. Delayed childbearing may lead to better economic standing, and hence lower mortality for men. Men who are older at the time of their first birth would have had more time during which to work and achieve more in the labor market without the need to provide for more dependents. However, the effects of age at

TABLE 5

HAZARD RATE MODELS FOR SURVIVAL PAST AGE 60, WIVES AND HUSBANDS MARRIED 1875–1899. INCLUDES CONTROLS FOR OCCUPATION. ENTRIES ARE COX PROPORTIONAL HAZARD REGRESSION COEFFICIENTS MULTIPLIED BY 10^3.

VARIABLE	WIVES MODEL 1	HUSBANDS MODEL 2
Age at first birth	2.62	−7.8*
Parity	15.1**	−0.42
Age at last birth	−10.5***	−9.25***
2-WAY INTERACTION		
Age at last birth * parity	−1.34**	−0.89
Occupation		
Nam-power occupational ranking	−1.60**	−2.03**
Farmer (= 1)	1.85	−4.39*
Occupation missing	40.2	114.9***
N	11140	11140
Δ -2LL	67.40	178.04
Δ df	12	12

*$p < 0.10$, **$p < 0.05$. ***$p < 0.01$

Adjusts for marriage year, LDS commitment, age difference between spouses, and number of children who died before age 18.

first birth on survival do not appear to be significantly different between husbands and wives (Models 5 and 6).

To address the possibility that socioeconomic status may account for the association between fertility and longevity, we incorporated husband's occupational status, measured from death certificates, into the analysis of both husband's and wife's longevity. The influence of fertility patterns on maternal and paternal longevity are not affected by the introduction of statistical controls for husband's occupation. Higher-status occupations are associated with lower mortality for both wives and husbands. Farmers had lower mortality rates ($p < 0.10$) than non-farming men. Men without an identified death certificate had significantly higher mortality rates. This suggests that these men were more mobile and likely died outside of Utah. Alternatively, these men may have been less socially integrated and information appearing on their death certificates was provided by less knowledgeable informants (i.e., non-kin). Men without linked death certificates were somewhat more likely to be non-Mormon and had wives who died at a somewhat earlier age than men with linked death certificates ($p < 0.05$ for simple differences). These factors would both reduce the chances of their death certificates linking to the UPDB and increase their chances of dying at a younger age.

DISCUSSION

Bearing and rearing children affects the mortality risks of post-reproductive mothers and fathers. Lower parity and late age at last birth are associated with

greater post-reproductive longevity among women. These associations are consistent with predictions based on evolutionary principles (hypotheses H2 and H3). These results also suggest that children receive resources from their parents to the detriment of the mother or that children with low-parity mothers are themselves low parity and are better able to provide assistance (hypothesis H5B). No evidence was found to support the prediction that late age at first birth enhances female longevity (rejecting hypotheses H1 and H4). Compared to their wives, husbands of these women experienced weaker longevity benefits associated with low parity and late fertility. Husbands lived longer lives when they fathered their first birth at older ages, but this effect was not statistically different from that of their wives. For the latter marriage cohort, when fertility rates began to decline, the reproductive histories of husbands and wives began to have statistically similar (and smaller) effects on their life spans, although wives' longevity continued to be more sensitive to the effects of parity and late fertility.

Our results suggest several interpretations. Women with many versus few children do not experience lower mid-life or late-life mortality as predicted by social support theories (House, Landis, and Umberson, 1988; Ross, Mirowsky, and Goldsteen, 1990). The economic benefits of large families during the latter half of the 1800s and access to informal social support through adult children (at least as measured by the number of children) do not appear to translate into longevity gains for post-reproductive parents. Children with many siblings may go on to have large families which in turn limits their availability to assist their aging mothers. The number of sons or daughters *per se* does not play a role in parental longevity (results not shown). Instead, high parity is associated with lower survival for post-reproductive mothers, an association consistent with theories of antagonistic pleiotropy and the disposable soma. That high-parity mothers live shorter lives also supports the idea that resources flow from parents to children (e.g., children are costly). It is possible that children must be co-resident or geographically close to their parents for there to be a detectable and salutary influence of parity on parental longevity. We were not yet able to consider this possibility at this time.

Late female fertility is associated with greater longevity after adjusting for age at first birth and parity. Evolutionary theories predict that later reproduction should be associated with greater longevity because it may be an indicator of a slower rate of aging. Further support for this interpretation is the finding that late fertile women under reproductive stress have lower mortality than other women. This latter result is important since women who age more slowly should be more resilient when faced with stress, such as childbearing and child rearing, than other women. As living conditions improved over time and the stress of reproduction declined, high parity that had previously differentiated the survival of late and non-late fertile women became less pronounced.

Fathers' survival is also related to reproductive history but not to the extent it is with mothers. The finding that men who fathered children late in life had longer lives is, to the best of our knowledge, new. It suggests that these men had slower age-related decline in fecundity than other men and likely had greater coital frequency. These reproductive changes may also be related to overall

rates of aging and may explain the better survival among late fertile men. This association may also reflect the fact that these older fathers were married to women much younger than themselves relative to other fathers. We know that as the age gap between husbands and wives increases the longer the life span of the husband; no effects in either direction have been detected for the wives. However, when we controlled for the age difference between husbands and wives, we found no change in the effect of husband's age at last birth on his longevity.

We have conducted further preliminary analyses that explore the familiality of both late fertility (Torgerson et al., 1997; Snieder et al., 1998) and longevity (Bocquet-Appel and Jakobi, 1990; McGue et al., 1993; Perls et al., 1998). Our analyses indicate that both brothers and sisters of late fertile women also age more slowly than siblings of non-late fertile women. This finding suggests that late fertility may be a marker for slow aging that is shared among relatives and that there are genetic variants in the human population that simultaneously slow aging, maintain female fertility later in life, and contribute to longevity in both sexes.

The novel results for husbands raise questions about social forces that are involved in the linkages between fertility and longevity. The effects of age at first and last birth on mortality risk among husbands and wives were shown to be statistically similar for the latter marriage cohort, although the impact of age at last birth was greater for wives and the influence of age at first birth larger for husbands. The point is that female reproductive history affects both spouses similarly, suggesting some environmental or marital mechanisms linking female fertility to either male or female mortality.

We considered the possibility that initially healthy couples go on to be more sexually active, have more children over more years, and also live longer. This argument suggests that enhanced fertility (high parity, low age at first birth, high age at last birth) should be associated with excess longevity because the initially healthy continue to be healthy through time, leading to both elevated fertility and longevity. In large part, this prediction is not supported in our analysis. Instead of detecting a positive association between high parity and excess longevity, we find an inverse relationship for mothers and no association for fathers. This selection argument would also predict that healthy persons should be better able to conceive earlier as well as to be longevous. Again, for women, age at first birth has no association with longevity, while for men young age at first birth is associated with higher mortality.

Some women may be capable of bearing children at advanced reproductive ages because they are initially healthy and especially robust. Perhaps late fertile women are more likely to possess some factor (possibly an innate robustness) that slows their rates of aging and enhances longevity. In other words, late fertility is not so much a result of initially high levels of robustness but an indicator of it. More work will need to be done to consider how lifestyle factors (e.g., diet, physical activity) may affect rates of aging, an important step in helping to understand gene-environment interactions.

Our analysis relies on selecting a sample of couples who survived to age 60 where the husband was not over 60 at the time of the last birth. These sample constraints mean that women who died as the immediate result of childbearing complications are excluded, as well as women

who died from other causes of death that arise in middle adulthood. We suggest that women who survived their childbearing years during the era analyzed here are more robust. This implies that our survivorship restriction is removing proportionately more women who are less resilient and perhaps aging more quickly. If this is the case, then women who have early ages at last birth (faster rates of aging) and who survive to age 60 are a hardier subset of all such women. This restriction would reduce the detectable mortality difference between late fertile and early fertile women. Men are less susceptible to this selection mechanism, at least as it pertains to the risks of childbearing, and this may help to explain why we see an effect of late fertility for men only during the latter marriage cohort.

It will be instructive to consider more recent marriage cohorts since fertility and mortality behavior has changed dramatically during the twentieth century. It will be important to follow longevity patterns, for example, among current cohorts where a proportion of women are delaying childbearing and thus having lower parity and later ages at last birth (Doblhammer, 2000). Also, further study is warranted about whether the health benefits of low parity persist or whether they are offset by the loss of access to adult children in later years. Analyzing the association between late fertility and age at natural menopause and their joint effects on female longevity will also be helpful. We are currently coding all causes of death for Utah decedents from 1904 to the present. When this work is completed, we will be able to assess whether certain reproductive patterns are associated with major causes of death (heart disease, cancer) where exposure to endogenous female hormones plays a role (Jacobsen et al., 1999; Kelsey and Bernstein, 1996).

We encourage further examination of the linkages between childbearing and parental mortality, particularly in the light of both sociological and evolutionary perspectives. Our analysis did not come down squarely in favor of one set of explanations over another, but we feel that considering both sets of predictions aided in enhancing our interpretation of the results.

ACKNOWLEDGMENTS

We wish to thank the Pedigree and Population Resource of the Huntsman Cancer Foundation, University of Utah, for providing the data and valuable computing support. This work was also supported by NIH grants AG13748 (Kinship and Socio-Demographic Determinants of Mortality, Smith PI) and AG14495 (Selection of Families for Genetic Analyses of Longevity; Cawthon PI). Helpful comments were provided on earlier drafts of this paper by Robert Mare, Myron Guttman, Cathleen Zick, Richard Cawthon, Nicholas Wolfinger, and Population Studies Seminar participants at the University of Utah. We also want to thank Diana Lane and Alison Fraser, who were instrumental in managing many of the complex data enhancements for this project.

REFERENCES

ANDERTON, D. L., and L. L. BEAN. 1985. Birth spacing and fertility limitation: A behavioral analysis of a nineteenth century frontier population. Demography **22:**169–183.

ANDERTON, D. L., NORIKO O. TSUYA, LEE L. BEAN, and GERALDINE P. MINEAU. 1987. Intergenerational transmission of relative fertility and life course patterns. Demography **24:**467–480.

ARRINGTON, L. 1958. Great basin kingdom: Economic history of the Latter-day Saints, 1830–1900. Lincoln, NE: University of Nebraska Press, p. 238.

ANTONOVSKY, A. 1967. Social class, life expectancy and overall mortality. Milbank Memorial Fund Quarterly, Health and Society **45:**1–74.

BEAN, L. L., G. P. MINEAU, and D. L. ANDERTON. 1983. Residence and religious effects on declining family size: An historical analysis of the Utah population. Review of Religious Research **25:**91–101.

BEAN, L. L., G. P. MINEAU, Y-C HSUEH, and D. L. ANDERTON. 1987. The fertility effects of marriage patterns in a frontier American population. Historical Methods **20:**161–172

BEAN, L. L., G. P. MINEAU, and D. L. ANDERTON. 1990. Fertility change on the American frontier: Adaptation and innovation. Berkeley, CA: University of California Press.

BEARD, C. M., V. FUSTER, and J. F. ANNEGARS. 1984. Reproductive history in women with coronary heart disease: A case-control study. American Journal of Epidemiology **120:**108–114.

BEETON, M., G. U. YULE, and K. PEARSON. 1900. Data for the problem of evolution in man, V; On the correlation between duration of life and number of offspring. Proceedings of the Royal Society **67:**159–179.

BELL, A. G. 1918. The duration of life and conditions associated with longevity: A study of the Hyde genealogy. Washington, D.C.: Genealogical Record Office.

BIDEAU, A. 1986. Fécondité et mortalité après 45 ans: L'aport des recherches en démographie historique (Fertility and mortality at ages 45 and over. The contribution of research in historical demography). Population **41:**59–72.

BITTLES, A. H., W. M. MASON, J. GREENE, and N. A. RAO. 1991. Reproductive behavior and health in consanguineous marriages. Science **252:**789–794.

BOCQUET-APPEL, J. P., and L. JAKOBI. 1990. Familial transmission of longevity. Ann. Hum. Biol. **17:**81–95.

BUSH, L. E. 1993. Health and medicine among the Latter-day Saints. New York: Crossroad.

CARNES, B. A., and S. J. OLSHANSKY. 1993. Evolutionary perspectives on human senescence. Population and Development Review **19:**793–806.

COOPER, G. S., and D. P. SANDLER. 1998. Age at natural menopause and mortality. Annals of Epidemiology **8:**229–235.

DOBLHAMMER, G. 2000. Reproductive history and mortality later in life: A comparative study of England and Wales and Austria. Population Studies **54:**169–176.

DORN, H. F., and A. J. MCDOWELL. 1939. The relationship of fertility and longevity. American Sociological Review **4:**234–246.

EGAN, K. M., E. GIOVANUCCI, L. TITUS-ERNSTOFF, P. NEWCOMB, and M. STAMPFER. 1997. Mixed blessing for middle-aged mothers. Nature **389:**922.

ENSTROM, J. E. 1978. Cancer and total mortality among active Mormons. Cancer **42:**1943–1951.

ENSTROM, J. E. 1989. Health practices and cancer mortality among active California Mormons. Journal of the National Cancer Institute **81:**1807–1814.

FREEMAN, B. C. 1935. Fertility and longevity in married women dying after the end of the reproductive period. Human Biology **7:**392–418.

FRIEDLANDER, NANCYLEE J. 1996. The relation of lifetime reproduction to survivorship in women and men: A prospective study. American Journal of Human Biology **8:**771–783.

GUO, G. 1993. Use of sibling data to estimate family mortality effects in Guatemala. Demography **30:**15–32.

GUO, G., and L. M. GRUMMER-STRAWN. 1993. Child mortality among twins in less developed countries. Population Studies **47:**495–510.

HAMILTON, W. D. 1966. The moulding of senescence by natural selection. Journal of Theoretical Biology **12:**12–45.

HOFFERTH, S. L. 1984. Long-term economic consequences for women of delayed childbearing and reduced family size. Demography **21:**141–155.

HOGAN, D. P., D. J. EGGEBEEN, and C. C. CLOGG. 1993. The structure of intergenerational exchanges in American families. American Journal of Sociology **98:**1428–1458.

HOLT, J. D., and R. L. PRENTICE. 1974. Survival analysis in twin studies and matched pair experiments. Biometrika **61:**17–30.

HOUSE J. S., K. R. LANDIS, and D. UMBERSON. 1988. Social relationships and health. Science **241:**540–545.

HUSTER, W. J., R. BROOKMEYER, and S. G. SELF. 1989. Modeling paired survival data with covariates. Biometrics **45:**145–156.

JACOBSEN B. K., S. F. KNUTSEN, and G. E. FRASER. 1999. Age at natural menopause and total mortality and mortality from ischemic heart disease: The Adventist Health Study. Journal of Clinical Epidemiology **52**(4):303–307.

KALBFLEISCH, D. J., and R. L. PRENTICE. 1980. The statistical analysis of failure time data. New York: Wiley.

KAPLAN, H. 1994. Evolutionary and wealth flows theories of fertility: Empirical tests and new models. Population and Development Review **20:**753–791.

KELSEY, J. L., and L. BERNSTEIN. 1996. Epidemiology and prevention of breast cancer. Annual Review of Public Health **17:**47–67.

KIRKWOOD, T. B. L. 1977. Evolution of aging. Nature **270**(5635):301–304.

KIRKWOOD, T. B. L., and M. R. ROSE. 1991. Evolution of senescence: Late survival sacrificed for reproduction. Philosophical Translations of the Royal Society of London, Series B, Biological Science **332:**15–24.

KITAGAWA, EVELYN M., and PHILIP M. HAUSER. 1973. Differential mortality in the United States: A study in socioeconomic epidemiology. Cambridge, MA: Harvard University Press.

LE BOURG, E., B. THON, J. LÉGARÉ, B. DESJARDINS, and H. CHARBONNEAU. 1993. Reproductive life of French-Canadians in the 17–18th centuries: A search for a tradeoff between early fecundity

and longevity. Experimental Gerontology **28:**217–232.

LEE, R. D. 1997. Intergenerational relations and the elderly. *In* K. Wachter and C. Finch (eds.), Between Zeus and the Salmon: The Biodemography of Longevity, pp. 212–233. Washington D.C.: National Academy Press.

LOGAN, J. R., and G. D. SPITZE. 1996. Family ties: Enduring relations between parents and their grown children. Philadelphia: Temple University Press.

LUND, E., E. ARNESEN, and J. K. BORGAN. 1990. Pattern of childbearing and mortality in married women—a national prospective study from Norway. Journal of Epidemiology and Community Health **44**(3):237–240.

LYCETT, J. E., R. I. M. DUNBAR, and E. VOLAND. 2000. Longevity and the costs of reproduction in a historical human population. Proceedings of the Royal Society of London B. **267:**31–35.

LYE, D. N. 1996. Adult child parent relationships. Annual Review of Sociology **22:**79–102.

MCGUE, M., J. W. VAUPEL, N. HOLM, and B. HARVALD. 1993. Longevity is moderately heritable in a sample of Danish twins born 1870–1880. J. Gerontol. **48:**B237–244.

MACINTYRE, S. 1997. The Black Report and beyond: What are the issues? Social Science and Medicine **44**(6):723–745.

MINEAU, GERALDINE PAGE. 1980. Fertility on the frontier: An analysis of the nineteenth century Utah population. Doctoral Dissertation. Dept. of Sociology, University of Utah.

MORGAN, S. PHILIP. 1991. Late nineteenth- and early twentieth-century childlessness. American Journal of Sociology **97:**779–807.

NAM, CHARLES B., and MARY G. POWERS. 1983. The socioeconomic approach to status measurement. Houston: Cap and Gown Press.

NATIONAL RESEARCH COUNCIL. 1989. Contraception and reproduction: Health consequences or women and children in the developing world. Washington D.C.: National Academy Press.

OAKES, D. 1986. Semi-parametric inference in a model for association in bivariate survival data. Biometrics **73:**353–361.

PEARLIN, L., C. S. ANESHENSEL, J. T. MULLAN, and C. WHITLACH. 1995. Caregiving and its social support. *In* R. H. Binstock and L. K. George (eds.), Handbook of Aging and the Social Sciences (Fourth Edition), pp. 283–302. New York: Academic.

PERLS, T. T., L. ALPERT, and R. C. FRETTS. 1997. Middle-aged mothers live longer. Nature **389:**133.

PERLS, T. T., E. BUBRICK, C. G. WAGER, J. VIJG, and L. KRUGLYAK. 1998. Siblings of centenarians live longer. The Lancet **351:**1560.

POWYS, A. O. 1905. Data for the problem of evolution in man. On fertility, duration of life and reproductive selection. Biometrika **4:**233–285.

RAHMAN, O. 1999. Family matters: The impact of kin on the mortality of the elderly in rural Bangladesh. Population Studies **52:**227–235.

ROSE, M. R. 1997. Toward an evolutionary demography. *In* K. Wachter and C. Finch (eds.), Between Zeus and the Salmon: The Biodemography of Longevity, pp. 96–107. Washington D.C.: National Academy Press.

ROSS, C. E., J. MIROWSKY, and K. GOLDSTEEN. 1990. The impact of the family on health. Journal of Marriage and the Family **52:**1059–1078.

SAMUELSSON, G., and O. DEHLIN. 1993. Family network and mortality: Survival chances through the lifespan of an entire age cohort. International Journal of Aging and Human Development **37:**277–295.

SILVERSTEIN, M., and V. L. BENGSTON. 1991. Do close parent-child relations reduce the mortality risk of older parents? Journal of Health and Social Behavior **32:**382–395.

SMITH, K. R., and C. D. ZICK. 1994. Linked lives, dependent demise?: Shared mortality of husbands and wives. Demography **31:**81–93.

SMITH, K. R., and NORMAN J. WAITZMAN. 1994. Double jeopardy: Interaction effects of marital and poverty status on the risk of mortality. Demography **31**(3):487–507.

SMITH, K. R., G. P. MINEAU, G. GUO, and D. HUTH. 1995. Fertility, marital and historical period effects on mortality risks of husbands and wives: Couple survival models for deaths 1840–1960 from the Utah genealogies. Paper presented to the Population Association of America, San Francisco, CA.

SMITH, K. R., and S. A. MCCLEAN. 1998. An introduction to paired hazard rate models in studies of the family. Journal of Marriage and the Family **60:**243–257.

SNIEDER, H., A. J. MACGREGOR, and T. D. SPECTOR. 1998. Genes control the cessation of a woman's reproductive life: A twin study of hysterectomy and age at menopause. J. Clin. Endocrinol. Metab. **83:**1875–1880.

SNOWDON, D. A, R. L. KANE, W. L. BEESON, G. L. BURKE, J. M. SPRAFKA, J. POTTER, H. ISO, D. R. JACOBS JR., and R. L. PHILLIPS. 1989. Is early natural menopause a biologic marker of health and aging? American Journal of Public Health **79:**709–714.

TORGERSON, D. J., R. E. THOMAS, and D. M. REID. 1997. Mothers and daughters menopausal ages: Is there a link? Eur. J. Obstet. Gynecol. Reprod. Biol. **74:**63–66.

TOWNSEND, PETER, NICK DAVIDSON, and MARGARET WHITEHEAD. 1988. Inequalities in health: The Black Report/The health divide. London: Penguin.

VAUPEL, JAMES W., JAMES R. CAREY, KAARE CHRISTENSEN, THOMAS E. JOHNSON, ANATOLI I. YASHIN, NIELS V. HOLM, IVAN A. IACHINE, VÄINÖ KANNISTO, AZIZ A. KHAZAELI, PABLO LIEDO, VALTER D. LONGO, YI ZENG, KENNETH G. MANTON, and JAMES W. CURTSINGER. 1998. Biodemographic trajectories of longevity. Science **280:**855–860.

WACHTER, K. 1997. Between Zeus and the salmon: Introduction. *In* Wachter, K. and C. Finch (eds.),

Between Zeus and the Salmon: The Biodemography of Longevity, pp. 1–16. Washington D.C.: National Academy Press.

WACHTER, K., and C. FINCH (eds.). 1997. Between Zeus and the salmon: The biodemography of longevity. Washington D.C.: National Academy Press.

WALDRON, I., C. C. WEISS, and M. E. HUGHES. 1998. Effects of multiple roles on women's health. Journal of Health and Social Behavior **39:**216–236.

WESTENDORF, R. G. J., and T. B. L. KIRKWOOD. 1998. Human longevity at the cost of reproductive success. Nature **396:**743–746.

WILD, C. J. 1983. Failure time models with matched data. Biometrika **70:**633–641.

WILLIAMS, G. C. 1957. Pleiotropy, natural selection, and the evolution of senescence. Evolution **11:**398–411.

WOLF, D. A. 1994. The elderly and their kin: Patterns of availability and access. *In* L. G. Martin and S. H. Preston (eds.), Demography of Aging, pp. 146–194. Washington, D.C.: National Academy Press.